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CardioPulse

Global Spotlights

How to diagnose and treat venous congestion in heart failure

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Heart failure (HF) is a clinical syndrome characterized by renal retention of salt and water due to cardiac dysfunction.[1] About 70% of the blood volume is contained in the systemic venous circulation. Due to their thin walls and high compliance, veins may dilate and accommodate large increases in blood volume with little or no increase in intravascular pressure. However, eventually, once the buffering capacity of the systemic venous system is exhausted, venous pressure will rise, leading to an increase in atrial and left ventricular filling pressures and wall stress and enhanced secretion of natriuretic peptides that should promote salt and water excretion, thereby limiting or reversing congestion. When the natriuretic peptide system is overwhelmed, congestion worsens, and the syndrome of HF develops. The rise in systemic venous pressure leads to increased transudation of water into the tissues, and when that exceeds lymphatic clearance capacity, organs and tissues will become oedematous, and symptoms and signs will develop. An increase in pulmonary venous pressure will lead to similar problems for the lung.

Thus, a diagnosis of HF can be made with confidence when there is evidence of cardiac dysfunction associated with a rise in natriuretic peptides and evidence of pulmonary and/or systemic venous congestion.[1] Echocardiography can easily identify, or exclude, the presence of structural or functional cardiac abnormalities; the assessment of early, pathological increases in circulatory volume is more difficult.

Most patients with HF are elderly: many might not exert themselves enough to become breathless or learn to avoid exercise that makes them breathless. They, or their physicians, may attribute breathlessness and inactivity to old age or comorbid lung or joint disease. Many older people have ankle swelling, but it does not necessarily reflect venous hypertension due to cardiac dysfunction. Alternative common causes include dihydropyridine calcium antagonists, alpha blockers and thiazolidinediones, venous insufficiency, or cellulitis. Empirical use of loop diuretics to manage oedema is widespread. For patients with congestion, diuretics might decrease blood volume and improve symptoms but potentially delay identification of HF.[2] For patients who are not congested, diuretics might activate neuroendocrine systems with deleterious consequences.

How to diagnose venous congestion in heart failure

A distended jugular vein usually indicates expanded intravascular volume and venous hypertension, but this clinical sign is often difficult to elicit reliably, particularly for non-specialists or in obese patients. Low serum albumin or haemoglobin concentrations might indicate plasma volume expansion, but these findings are also common in the presence of malnutrition, liver, bowel, or kidney disease. Several formulae have been proposed to estimate plasma volume non-invasively based on haematocrit and body weight, but their clinical value is dubious.[3]

In summary, of the many surrogate measures of congestion, none is sufficiently specific or sensitive to provide robust support for a diagnosis of HF.

In expert hands, the inferior vena cava (IVC) is commonly visualized using ultrasound from the subcostal view: measuring its diameter and respiratory variations allows the estimation of intravascular volume. In a small mechanistic trial, a 48 hours suspension of medical therapy in ambulatory patients with mild symptoms of HF was associated with a 20% increase in IVC diameter and doubling of plasma N-terminal prohormone of brain natriuretic peptide (NT-proBNP), without substantial changes in body weight. In patients with acute HF, serial IVC assessments provide useful information to monitor response to decongestive therapies and identify, prior to discharge, those at greater risk of death or re-hospitalization.[4]

When the IVC is ≤16 mm in diameter and collapses further with inspiration, this suggests that congestion is absent; a distended IVC with loss of inspiratory collapse indicates congestion and a high risk of cardiovascular events.[5] Imaging the heart by a subcostal view permits diagnosis of pleural and pericardial effusion and ascites, suggesting either a local pathology (inflammation or cancer) or HF. Therefore, the IVC should be assessed routinely in those with suspected or established HF undergoing echocardiography (*Figure 1*).

When the IVC is not identifiable, intravascular volume can still be estimated by placing a high-frequency ultrasound probe over the neck of the patient, to measure the internal jugular vein diameter or its cross-sectional area and evaluate its response to a Valsalva manoeuvre. A

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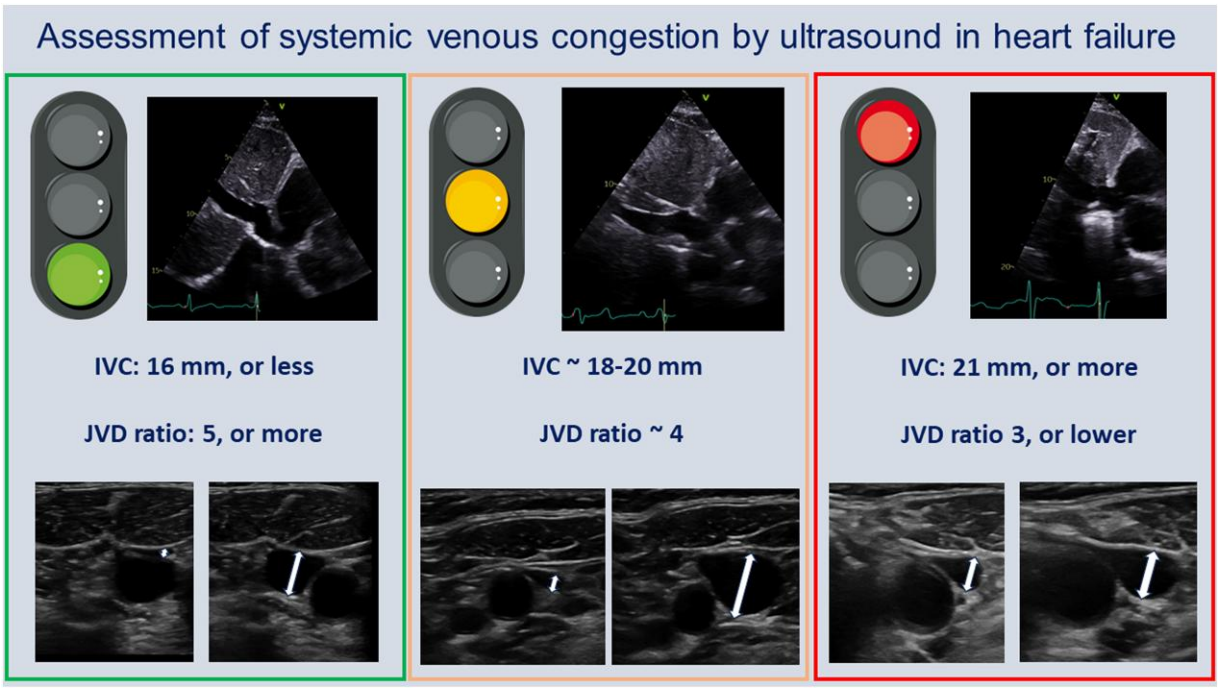
Assessment of systemic venous congestion by ultrasound in heart failure
IVC: 16 mm, or less
JVD ratio: 5, or more
IVC ~ 18-20 mm
JVD ratio ~ 4
IVC: 21 mm, or more
JVD ratio 3, or lower

Figure 1 With increasing severity of systemic venous congestion (left to right), the inferior vena cava dilates. Systemic venous congestion by ultrasound can be also assessed by calculating the ratio between the diameter of the jugular vein during Valsalva to that at rest. Normally, this ratio is >4, but when intravascular volume expansion develops, the internal jugular vein at rest is distended, and due to a limited compliance, it will not expand more than 2–3-fold during a Valsalva manoeuvre (bottom right). The image has been created using Servier Medical Art (used with permission under a Creative Commons CC BY licence).

distended internal jugular vein that does not increase further in size during Valsalva indicates a raised right atrial pressure and intravascular volume overload and the need to intervene to reduce congestion.[6] Combining the assessment of IVC with that of portal and renal veins provides additional diagnostic and prognostic information.[4] The IVC or jugular vein diameter are not greatly influenced by left ventricular ejection fraction (LVEF) or measures of body size.[5,6] However, in healthy, young individuals a dilated IVC can reflect the effects of intense training and/or low venous tone. Right heart catheterization is considered the gold standard to assess pressure and volume congestion, but due to its invasive nature and need for specialist equipment and expertise, it is reserved for cases where diagnostic doubt persists despite non-invasive investigation or as part of a workup for those in need of therapies for advanced disease. Several other non-invasive technologies, such as near-infrared spectroscopy or bioimpedance, can be used to estimate intravascular volume.[7] Implantable sensors have also been developed to monitor IVC size as well as pulmonary artery or left atrial pressure.[8]

How to treat venous congestion in heart failure

Loop diuretic agents have a class I recommendation in European Society of Cardiology (ESC) guidelines for the treatment of congestion, but as noted above, the clinical evaluation of congestion is largely subjective. Intravenous loop diuretics are probably life-saving in patients with acute pulmonary oedema and may improve symptoms and signs for those with peripheral oedema. However, loop diuretics are not known to increase survival in chronic HF and might enhance renin–angiotensin system activation. Optimization of therapy is essential to avoid administration of loop diuretics to those who do not need them and to prevent recurrence of congestion and improve outcomes for those who do. Mineralocorticoid receptor antagonists have modest diuretic effects but improve prognosis in patients with reduced LVEF [heart failure with reduced ejection fraction (HFrEF)] and probably also in those with a higher LVEF. In patients resistant to a loop diuretic, adding a thiazide or a sodium–glucose cotransporter 2 inhibitor can potentiate natriuresis and diuresis. Acetazolamide might also be effective in the short term.[9] In selected cases, structural valve interventions may be required to improve management of severe congestion.

However, management of venous congestion is not a one-size-fits-all endeavour, as each patient's journey is unique. Empowering patients with knowledge about their condition and care plan fosters compliance and self-management. Patients should understand the importance of adhering to dietary guidelines, including avoiding excessive amounts of alcohol, sodium, and fluid intake.

Future directions

Several trials are now testing the hypothesis that treatment guided by invasive or non-invasive assessment of IVC can improve the management of congestion in patients with HF (NCT04203576, NCT05090137, NCT05763407). New technologies are also being developed that modulate IVC flow to decrease renal venous congestion and, hopefully, improve diuresis in the most severe HF cases (NCT03234647). Whether screening for evidence of venous congestion in its early stages is clinically useful is uncertain, but, when detected, it would

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seem wise to re-double efforts to optimize control of hypertension and other cardiovascular risk factors: pre-clinical intravascular congestion may be common (~20%) in people with or at high risk of cardiovascular disease.[10] Identifying, quantifying, and managing venous congestion with more precision might avoid the deleterious consequences of inappropriate diuretic therapy as well as delaying or preventing the onset of symptomatic HF: this will be a crucial focus for research in the coming decade.

Declarations

Disclosure of Interest

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